Modern Concepts of Cardiovascular Disease

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THE RELATION BETWEEN THE HEART AND THE KIDNEYS

I. Physiological Considerations

It must be of some significance that in man and in the larger mammals in general the weight of the heart equals or exceeds the combined weight of the kidneys. This even holds for the whale[1]. It is tempting to relate this fact to the more striking fact that some 1300 cc. of blood, more than one-fourth of the basal cardiac output, flows through the kidneys of the average adult male per minute[2]. It is no wonder, therefore, that under ordinary conditions it is difficult to produce any remarkable increase in renal blood flow, although the reverse process occurs from a variety of causes.

The reason for the enormous renal blood flow is, presumably, the high degree of development of the glomerular kidney, which was originally evolved in marine forms when the ocean was not yet salty. The vicissitudes of the glomeruli in the kidneys of salt water fish furnish a fascinating chapter in comparative morphology and physiology[3]. Aglomerular kidneys developed in some species. Among the land-living vertebrates, birds and mammals acquired the loop of Henle[4] for the preservation of body water and the production of a urine more concentrated than the blood in many respects.

The added element of high blood pressure in the glomerularized kidney has undoubtedly affected the work and size of the heart to an important degree. How the kidney, more specifically the glomeruli, keeps the central pump or its nervous regulators informed of the requirement of blood flow and pressure for the kidney is unknown. The remarkable response of the general arterial pressure to partial constriction of the renal arteries, as in the Goldblatt animal[5], necessitates a mechanism within the kidneys for the production of a pressor hormone. Does the kidney normally play a role in the regulation of arterial pressure? The recent histological studies of Goormaghtigh[6] seem to indicate the existence of specialized secretory cells in the wall of the afferent glomerular arteriole which multiply and presumably hyperfunction in the Goldblatt kidney.

Apart from these interrelations between the kidneys and the heart in which the kidneys are dependent upon the work and energy of the heart, there is the important opposite situation in which the circulation must have the cooperation of the kidneys. I am referring here to the role of the healthy kidney in maintaining a normal plasma volume and a normal colloid osmotic balance between the vascular and extravascular or interstitial fluids. Sudden or prolonged rises in circulating plasma volume after the rapid ingestion or injection of fluids are prevented by the diuretic response of the kidneys. Similarly, decrease in plasma volume is rapidly reflected in oliguria. Of equal importance is the practical non-permeability of the normal glomerular membrane to plasma protein. Were it not for this, we would all have the nephrotic syndrome. However, even the glomerular membrane is dependent upon an adequate, properly oxygenated, renal arterial blood flow for normal resistance to the escape of plasma protein.

The role of the kidneys in maintaining relative and optimal constancy of the internal fluid environment by the excretion of the organic and inorganic ashes of the internal metabolic fire and by the reabsorption of glucose, water, necessary electrolytes and vitamines requires no elaboration. Suffice it to say that the tubular function of reabsorption is beautifully adjusted to the needs of the body as a whole[7], and, probably, to the needs of the most active muscle in the body, the heart.

II. Pathological Physiology

This will be considered from the standpoint of (a) primary disease or dysfunction of the heart, and (b) primary disease or dysfunction of the kidneys.

(a) Cardiac disease affects the kidneys largely by way of central circulatory failure or weakness of the pump. Theoretically, the result may be decreased renal arterial blood flow, venous congestion, or both. The effects of passive congestion on the kidneys are recognized, but the demonstration of decreased renal blood flow in heart failure is not so obvious. Judging from the urea clearance there is often little change in renal function, in spite of oliguria and other signs of renal circulatory disturbance[8]. More work must be carried out on patients using the newer methods designed to measure total renal blood flow and active tubular mass[9]. In general, heart failure is of relatively little significance insofar as renal function is concerned unless it is superimposed upon pre-existing diffuse renal disease, making a combination dangerous to renal function and often to the life of the individual. Finally, congestive heart failure may cause sufficient albuminuria to result in depletion of plasma proteins and the vicious synergism of nephrotic and cardiac edema.

Another well-known effect of heart disease upon the kidneys consists of the partly mechanical, partly toxic focal alterations due to multiple embolization or thrombosis of the terminal renal vessels in various types of endocarditis. Also, bacteremia may lead to the development of diffuse glomerulonephritis in this group.

(b) Kidney diseases may react upon the heart in several different ways: In acute nephritis (1) by causing oliguria or anuria, secondary increase in blood volume and at least temporary increase in the load upon the heart. On the whole, this is an unimportant effect unless the fluid intake is excessive. More serious are (2) the mechanical embarrassment of the heart by acute pericardial effusion, hydrothorax and ascites, and (3) the increased work of the heart resulting from sudden rise in arterial pressure, especially when the latter is part of attacks of acute hypertensive encephalopathy with convulsions. Left ventricular failure may occur rapidly under these conditions and produce fatal pulmonary edema.

In chronic renal disease, the chief damage to the

heart results from several factors: (1) Persistent hypertension, due to a general increase in the peripheral resistance, leads to cardiac hypertrophy, eventual dilatation and failure. (2) Anemia in chronic renal insufficiency cannot be an indifferent item to the heart, either from the standpoint of cardiac work or the direct effect of deficient blood upon the myocardium. The resistance of this type of anemia to treatment makes the cardiac problem all the more serious. (3) Renal insufficiency in the preuremic stage, at a level of one-third or less of the average normal renal function, seems to have a deleterious effect upon the heart, particularly when superimposed, as is often the case, on hypertension and anemia. (4) Uremic pericarditis is chiefly of pathological interest, as it seems to produce no special effects upon the function of the heart unless considerable pericardial effusion occurs.

III. Clinical Applications of Cardiorenal Interrelations

Several clinical deductions may be drawn: (1) The common role of the heart and the kidneys in relation to the causation and removal of edema makes it imperative that we distinguish accurately between cardiac and renal aspects of edema. The success of powerful diuretics depends upon their excretion by the kidneys and hence on the level of renal function. It is, therefore, unwise to use these agents unless kidney function has been estimated. However, in case of doubt as to the cause of the edema, it is always safe to digitalize the patient, since digitalis does not seem to depend upon good renal function for its excretion. The edema in the hypertensive forms of chronic renal disease is likely to be cardiac in origin. The edema of non-hypertensive renal disease is rarely cardiac. Albuminuric or nutritional hypoproteinemia should be thought of in patients with persistent massive cardiac edema who have little dyspnea or venous congestion after adequate digitalization and restriction of salt and fluids.

(2) Oliguria may be either cardiac or renal in origin but, with very rare exceptions, anuria is renal. In the attempt to restore urinary flow, due consideration must be given to the tolerance of the heart for intravenous fluids, in particular for strongly hypertonic solutions. Anuric kidneys do not benefit from cardiac failure induced by overhydration.

(3) Hypertension is perhaps more dangerous in acute than in chronic renal disease. Left ventricular weakness or failure is commonly overlooked in acute nephritis. Instead of the uncontrolled use of diuretics of hypertonic solutions in these cases, immediate relief of the left heart by means of venesection, oxygen, morphine and digitalis is indicated. The symptoms and signs of right heart or congestive failure should perhaps be eliminated from our text-books and lectures until a generation of physicians has grown up fully aware of the symptoms and signs of early left ventricular failure.

In chronic hypertensive renal disease major attention must be centered on maintenance of good cardiac function, rather than on the more or less ceremonial attempts at dietary treatment of the renal condition. The lower the renal function, the more the patient's life depends on the efficiency of his heart. The common practice of "flushing out" the kidneys with large amounts of fluid may lead to embarrassment of the circulation as a major consequence, and to nocturia and disturbed sleep as a minor irritation. A daily urine volume of 1500-2000 cc. is sufficient for all degrees of renal insufficiency, even on a normal diet. The earliest signs of left ventricular weakness should be the signal for digitalization and other measures of cardiac therapy. Every patient with essential hypertension should be regarded as a candidate for later heart disease.

(4) Anemia in the later stages of renal disease is caused, presumably, by a toxic depression of the bone marrow and ultimately requires blood transfusion. Hypertension and anemia are a bad combination for the heart, even in the absence of renal insufficiency. Unusual care must be exercised in carrying out blood transfusions in patients with low renal function to prevent the fatal effect of incompatible blood and the common danger of pulmonary congestion or cardiac failure, due to excessive increase in blood volume in patients who already have some circulatory insufficiency. Only small transfusions are safe, and with sufficient interval between transfusions to allow for hemodynamic adjustments. One should be satisfied with a hemoglobin level of 75 or 80 per cent.

IV. Summary

The heart and the kidneys stand in intimate physiological relationship because of the peculiar demands of renal function in the mammalian kidney for a large renal blood flow as well as for high glomerular capillary blood pressure. In turn, the kidneys help to maintain a constant plasma composition so necessary in the nutrition of tissues and in the dynamics of fluid exchange between the vascular and extravascular spaces. The kidney develops a chemical mechanism for the regulation of blood pressure when its circulation is restricted; perhaps, also, under normal circumstances. The implications of such a mechanism on the function of the heart are interesting to consider.

In disease of the heart, the kidney is involved usually by way of passive congestion, without serious consequence ordinarily. However, in circulatory shock severe renal insufficiency may develop, rarely in previously normal kidneys. A nephrotic edema may be superimposed on original cardiac edema because of excessive proteinuria or undernutrition. The mechanical effect of emboli on the kidneys is obvious and chiefly of diagnostic importance. In general, the fate of the primarily cardiac patient is not seriously dependent upon his kidneys, except in old age when renal arteriosclerotic atrophy supervenes.

However, in primary disease of the kidneys, the status of the heart is often decisive. In acute nephritis, left ventricular failure and pulmonary edema may rapidly terminate the disease fatally. In chronic renal disease, whenever hypertension occurs, potential and later actual cardiac insufficiency become a primary concern in the patient's management. Failure of the heart often precipitates severe renal insufficiency in these cases. The harmful effects of anemia and of uremia upon the heart have been considered.

A better understanding of the interrelations between the kidneys and the heart should be of value in establishing a more rational therapy.

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